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Predicting the Risk of Freezing the Skin

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Summary

It is well known that wind increases the risk of frostbite during exposure in a cold climate. The explanation is that increased airspeeds enhance heat transfer from the body. This effect was quantified by Siple and Passel in the 1940s (14). They measured the time needed for water, inside a cylinder, to freeze during exposure to various combinations of air speed and temperature. From these data, they developed the so-called windchill index (WCI) for predicting the heat transfer from nude body parts. Later WCI was also expressed as “equivalent temperature” (T_e). However, a reexamination of the Siple and Passel data has shown that WCI (and T_e) does not correctly describe the wind induced heat transfer (5). As charts based on WCI and T_e are frequently used to express cold weather severity these indices should be corrected. Another shortage is that important parameters for predicting windchill are limited to air speed and temperature. A previously presented skin-frostbite risk model (5) has been developed further, now also allowing simulation of wet skin and solar radiation. The model suggests that the risk for finger frostbite increases from 30 to 70 % after wetting the skin when the air speed and temperature is 6,8 m/s and -15°C, respectively. This prediction is similar to experimental results found in the literature. There is a common opinion that windchill skin injuries are rare in the Antarctic during the summer. It is estimated that solar radiation corresponds to 5 to 10 °C higher air temperature. These values are much the same as those suggested by the model at solar intensities common during the Antarctic summer. Another opinion is that time spent in cold weather regions reduces the risk of skin frostbite considerably. This adaptation has been found to reduce the risk of finger frostbite from 74% (1st year men) to 29% (2nd year men). Such adaptation means that twice as high air speed or 2 - 5 °C lower air temperature is allowed at unchanged risk of skin injury according to the model. Risk model predictions have also been compared with cold weather injuries among U.S. soldiers in Alaska. It was found that the equivalent temperatures (T_e) that were associated with the greatest change in cold weather injuries coincided fairly well with increased risk of frostbite according to the model whereas commonly used T_e -windchill chart seems to underestimate the risk.

Introduction

Low air temperatures and high wind speeds are associated with an increased risk of skin freezing. Such injuries may result in extensive loss of duty-time and can also require long medical treatment. Hence, tools for predicting the risk of skin frostbite is valuable in order to reduce cold weather casualties. Siple and Passel (1945) exposed bare skin to different climates and observed at what combinations of air speed and temperature skin freezing occurred. In addition, they performed cooling experiments on a water-filled cylinder from which they derived their windchill index (WCI).

$$WCI = 1,162 \cdot (10,45 + 10 \cdot v^{0,5} - v) \cdot (33 - T_a) \quad (W/m^2)$$

where v is the air speed (m/s) and T_a (°C) is the ambient temperature.

They reported that an increased risk of frostbite was prevalent at a WCI above 1400 ($kcal \cdot m^{-2} \cdot h^{-1}$). Finger frostbite at considerably lower WCI values than 1400 has also been reported. These exposures were, however associated with snow in the air or wet skin. Another factor of importance is presence of solar radiation and time spent in cold weather regions.

The use of the windchill index has been widely spread, but its foundations and interpretation have been questioned from time to time. Surveys conducted in Canada (11) showed that the knowledge of windchill differed considerably between regions and between groups of people. Generally, a windchill index expressed as equivalent temperature (T_e) was preferred before e.g. heat flux-value expressed in W/m^2 or a WCI without a unit. Yet, it was emphasised that informing the general public is a major challenge as windchill indices often are presented in units that are not understood or in units causing confusion (11).

Different types of "Equivalent temperature" (T_e) have been derived based on slightly different conditions (11).

$$T_e = 33 - WCI / [1,162 \cdot (10,45 + 10 \cdot v_{ref}^{0,5} - v_{ref})] \quad (^\circ C)$$

where WCI is the windchill and v_{ref} is the reference air speed (often 1,7 or 2,2 m/s). The most commonly used T_e originates from the WCI of Siple and Passel (14) where T_e is calculated using a "reference air speed", often 6 or 8 km/h. This apparently small difference give rise to a $5^\circ C$ difference in T_e , a difference that may cause a significant change in cold stress. If T_e is used only as an indication of thermal load this can be accepted. But the T_e - charts are often marked with risk zones informing of " Little risk", "Increasing risk" and "Great risk", zones where a $5^\circ C$ difference can mean safe exposure time ranging from hours to 1 minute. An individual or a military commander preparing for cold weather activities would probably prefer a more informative risk chart, also including other risk factors relevant for windchill skin injuries.

Although both the WCI and related T_e are popular and frequently used these hold some disadvantages. One of them is that the heat transfer coefficient is incorrect not taking consideration in the air speed contribution at higher wind. Other drawbacks are that WCI have no base in human physiology or body parts dimensions. Nor is WCI suitable for predictions including additional parameters such as solar radiation, wet skin, acclimation e.t.c. Until now e.g. the effect of solar radiation is taken into account by introducing a correction factor in the form of adding a T_e -value. However, such a procedure is not, in principle, advisable because it tends to hide the mechanisms involved.

Therefore, the aim of this work was to improve a previously presented windchill model for prediction of the risk of skin frostbite (5) by including the effect of wet skin and solar radiation. The new model is based on general physical relationships describing the heat flux and temperature distribution from the skin to the environment. The physical model is linked to a risk model based on human data presented in the literature.

Heat transfer mechanisms

Skin temperature is mainly a result of two factors, heat loss rate to the environment and rate of heat input from the blood. A falling skin temperature can be counteracted if the physiological reaction CIVD (cold-induced vaso-dilation) opens the vessels so warm blood can reach the skin. The blood flow capacity in e.g. the fingers is so great that most windchill conditions can be compensated for if the CIVD can act in time. But, skin blood flow is impossible if the blood has frozen, a situation that happens at a blood temperature around $-1^\circ C$ (8). So, for a given CIVD reaction time the heat transfer rate from the skin decides whether the skin will freeze or not.

Convective dry cooling. Before the hunting reflex starts, the heat production and blood heat transfer to the skin are normally so low that the temperature change in the skin mainly depends on heat content of the actual body part and heat transfer rate from the skin to the environment. This physical process is driven by the temperature difference between the skin and the environment. The heat resistance of the skin, from the depth of the skin blood vessels outwards, is constituted by the insulation of cutis and epidermis. However, more important to the heat transfer rate is the insulation of the apparent still air layer surrounding the body part. The thickness of this layer depends on the air flow and surface characteristics. A textile layer or even a short beard improves the thickness considerably, thus reducing the heat loss from the skin. Consequently less skin blood flow is needed.

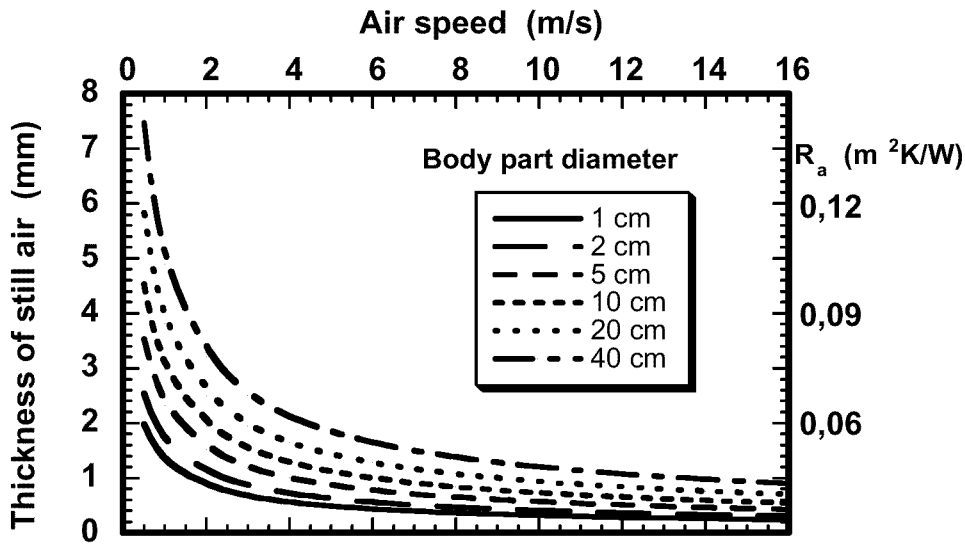


Figure 1. The thickness of the apparent still air layer (mm) surrounding an approximately circular body part in cross wind. The air layer is also given as insulation value ($\text{m}^2\text{K/W}$) including the radiation heat transfer coefficient.

The apparent still air layer thickness is calculated from the convective heat transfer coefficient (h_c) and it depends on a number of factors as body part dimension and shape, relative air speed and degree of external turbulence (fig. 1). If the wind flows across the body and the external air flow is approximately laminar the various body parts can be considered as cylinders of various shapes giving different heat transfer coefficients. The airflow characteristics around a circular cylinder depend strongly on the Reynolds number (Re) (no dimensions, ND).

$$Re = v \cdot d / \nu \quad (\text{ND})$$

where v is the air speed (m/s), d is the body part diameter (m) and ν is the kinematic viscosity (m^2/s). Hilpert (7) found that the average Nusselt number, Nu (ND), for a cylinder could be written as

$$Nu = h_c \cdot d / \lambda \quad (\text{ND})$$

where h_c ($\text{W} \cdot \text{m}^{-2} \cdot \text{K}^{-1}$) is the forced convection coefficient, λ ($\text{W} \cdot \text{m}^{-1} \cdot \text{K}^{-1}$) is the thermal conductivity of the surrounding medium and $Pr = \nu / \alpha$ is the Prandtl number (ND) where α is the thermal diffusivity (m^2/s).

Combining the equations above gives the general expression for h_c as

$$h_c = 4,47 \cdot d^{-0,38} \cdot v^{0,62} \quad (\text{W} \cdot \text{m}^{-2} \cdot \text{K}^{-1})$$

for an air temperature of -25°C where the coefficient decreases from 4,47 to 4,37 at 0°C . The equation obtained by Danielsson (3) for a standing human, measured at $+28^\circ\text{C}$ was

$$h_c = 3,76 \cdot d^{-0,36} \cdot v^{0,61} \quad (\text{W} \cdot \text{m}^{-2} \cdot \text{K}^{-1})$$

which is fairly similar to the correlation equation valid for cylinders in cross flow. The slightly lower coefficient for the human body depends somewhat on the higher temperature but mainly on the interference from adjacent body parts on the air streams (3). The formula displays the average h_c -value of the body part. If the heat transfer of a specific site is of interest the local h_c -value must be considered. It depends on the angle to the wind but is also affected by e.g. the interference from clothing items (4). The deviations around

the circumference of a body part can be fairly great compared with the average h_c -value (3). Undisturbed free air flow generally produce the greatest h_c -value on the windward side whereas the lowest values are found at right angle to the wind. On the leeward side there is another maximum but which is lower than on the windward side. For a part with approximately circular shape the h_c -value on the windward side is roughly 40% greater than the average value for all angles.

Even at strong winds the air layer, close to the body surface is approximately still. This layer constitutes a resistance against heat as well as mass transfer. Neglecting the long-wave radiation heat transfer at the skin surface, which is acceptable for slim body parts in "strong" winds, the thermal insulation value of the air layer, R_a , is calculated by inverting the h_c -value ($R_a = h_c^{-1}$). The result of such calculations, for different air speeds and body diameters, clearly shows that slim body parts are much more exposed to high cooling rates than wide ones. However, comparing these R_a -value with the insulation value of the cutis and epidermis, it becomes clear that the skin is of less importance than the air layer in protecting the deeper lying tissue. In the present model it is assumed that the thickness of the epidermis is 0,2 mm (normal range 0,1-0,7 mm) having a thermal conductivity of $0,21 \text{ W}\cdot\text{m}^{-1}\cdot\text{K}^{-1}$ whereas the cutis is set to be 1,5 mm thick (range 1-2 mm) with the conductivity $0,37 \text{ W}\cdot\text{m}^{-1}\cdot\text{K}^{-1}$ (6). Hence, the insulation value of the skin is approx. $0,006 \text{ m}^2\text{K/W}$. Even with a "thick skin" the air layer still dominates in respect of heat resistance. Nevertheless, the importance of the skin should not be underestimated as protection is a question of time until the hunting reflex is activated. So, even a small heat resistance value can reduce the temperature drop enough to make CIVD possible. Figure 1 clearly illustrates that great changes in air layer insulation occur at low air speeds whereas the relative effects are minor at strong winds. This explains why windchill charts normally don't cover air speeds greater than about 20 m/s. The shape of the R_a -curves also explains the importance of the reference air speed selected when calculating T_{cl} . The two reference air speeds, 6 or 8 km/h (1,7 and 2,2 m/s respectively), normally used differ about 5°C in T_{cl} , a difference that can be significant in terms of body cooling. So, the way of calculation is of importance when comparing T_{cl} -values.

Evaporative cooling In the physical skin model heat is conducted through the skin components (cutis and epidermis). Various thermal properties of the skin are allowed by e.g. introducing water in epidermis. Water in epidermis can be combined with evaporation or not. Evaporation is assumed to take place at the skin surface and the rate of mass transfer depends on the ambient air layer thickness. Here, the skin is assumed to be uncovered but e.g. the effect of textile layer can be included. Under normal conditions when forced convection is valid, turbulent or not, the evaporative heat transfer can be estimated from Lewis relation, L , where

$$L = h_e / h_c = 16,7 \quad (\text{K/kPa})$$

where $h_e (\text{W}\cdot\text{m}^{-2}\cdot\text{kPa}^{-1})$ is the evaporative heat transfer coefficient. The apparent still air layer in terms of thickness, impeding the transportation of heat and mass, is approximately the same for heat, $d_{th} = 0,26 / h_c$ (m) as for mass, $d_e = 0,031 / h_e$ (m). The evaporative heat transfer is assumed to be forced by the water vapour pressure difference between the ambient air and the skin surface. The vapour pressure of the air is calculated from the temperature assuming a relative humidity (rh) of 80%. The partial pressure at the skin is based on rh = 100% and a skin temperature around 0°C .

Solar radiation. A balancing factor to the convective cooling of the skin is absorption of solar radiation. Such an input impedes the skin temperature drop or may even result in skin temperature rise. The solar heat input can vary considerably, from almost none to a net input reaching almost 1000 W/m^2 . The short-wave radiation reaching the skin comes both directly and as reflection from the sky and the ground (albedo).

The direct radiation heat flux reaching the skin depends on the cloudiness, solar altitude and position of the exposed object. The sky- and in particular the ground albedo cause an heat inflow that is less dependant on angle between the incoming solar beam and the position of the object. This is because of the strong reflection if the ground is covered by fresh snow. For instance, if the face is turned away from the sun the direct solar radiation reaching the face is negligible whereas the radiation coming from the ground- and sky albedo can be of the same magnitude as the direct solar heat flux. When the sky is overcast, the albedo may become greater or much greater than the direct radiation because of multi-reflexion between the ground and the clouds (9). This situation demands, however, a very good reflectivity of the ground. If it is covered by fine,

fresh snow the reflectivity is very great, around 85%, whereas as comparison, a grass or soil surface reflection rarely exceeds 15-20%. The amount of solar radiation, absorbed by the nude skin depends slightly on e.g. the skin properties. The model assumes that 65% of the incoming short-wave radiation is absorbed whereas the long-wave emittance is set to 0,97. The radiation heat exchange occurs at the outermost layer of the skin both in case of absorption of short wave radiation or emittance of long-wave, thermal radiation. If there is a net inflow, the skin surface temperature rises, reducing the heat loss from deeper skin layers. The thermal effect of the solar radiation depends on the wind. At the same time as a higher skin temperature reduces the heat loss from deeper lying tissue, the heat loss outwards increases because of greater temperature difference. So, if the insulating air layer is thick, i.e. the air speed is low the effect of solar radiation becomes great. This is one reason why the clothing can be very light in Antarctic, in the summer. The temperature is rarely below -10°C , the wind is often light and there is a bright sunshine. However, if the wind is strong, reducing the air layer thickness, the advantage of sunshine becomes reduced as the absorbed heat is lost back to the environment to a higher degree.

Human windchill data

The physical model allows calculation of temperature gradients and heat fluxes if the boundary conditions are known. The model is not defined for specific heat transfer avenues. Hence, it could also be used for estimating the risk for burn injury. Irrespective of purpose, the model needs information on how the human body reacts on various climatic exposures. The technique used here, is to combine the physical model with the frostbite risk distribution associated with skin surface temperatures obtained from human studies.

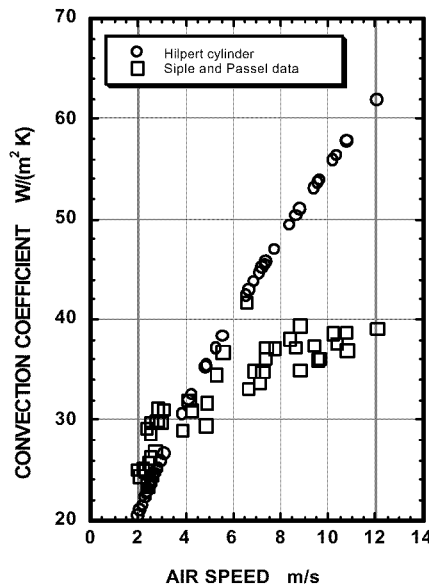


Figure 2. Comparison between convection coefficient suggested by Siple and Passel (14) and that normally obtained for a cylinder with the same diameter (6 cm) in cross flow (7).

Risk of frostbite. The Siple and Passel study (14) did not include human data, at first. Their experiments focused on the cooling rate of a water filled cylinder. They measured the time it took for the water to freeze for various combinations of air speed and temperature and from these data the cylinder heat flux-values (W/m^2) were calculated. However, the conversion from convective heat flux to "windchill factor" (convection heat transfer coefficient) included an error which still is present in all WCI and T_{e} -values. Siple and Passel did not account for the insulation of the cylinder wall. Consequently, their convection coefficient became incorrect and the relationship between wind speed and h_c differs from what is normal for cylinders in cross flow (figure 2). The consequence for the WCI and T_{e} -values is that these are based on a convection coefficient that is strongly underestimated, starting at air speeds exceeding 4-5 m/s (5).

Siple and Passel introduced the human aspect by exposing themselves and colleagues to different climates, noticing at which temperature and air speed frost nip occurred in the face. The corresponding windchill index was calculated from their windchill factor setting the skin temperature to 33°C. In spite of unphysiological skin temperature and a physically incorrect convection coefficient, yet Siple and Passel found that skin frostbite seldom occurred at windchill indices lower than 1400 ($\text{kcal} \cdot \text{h}^{-1} \cdot \text{m}^{-2}$). They also identified two other stages corresponding to WCI = 2000 and 2300. The "relative human comfort" for these three stages were verbalised as "Freezing of human skin begins, depending on activity, solar radiation, character of skin circulation. Travel and life in temporary shelter becomes disagreeable" (WCI=1400), "Conditions for travel and living in temporary shelter becomes dangerous. Exposed areas of face will freeze within less than one minute for the average individual" (WCI=2000) and "Exposed areas of face will freeze within less than half a minute for the average individual" (WCI=2300). These characterisations are still used in the widely spread WCI / T_e -tables.

Around 1970 Wilson and Goldman (16) conducted experiments on finger freezing in cold wind. They found almost no skin freezing at WCI values below 1400; values above this was often but not always associated with skin freezing. These data imply that a WCI of 1400 is a fairly good indicator of air speed and temperature that can cause nude skin freezing. However, they measured a considerable variation in the skin temperature when freezing occurred. Furthermore, their data on the freezing temperature of the skin differed considerably from those found by Keating and Cannon (8) who suggested a temperature around -1°C. Wilson and Goldman (16) and Wilson et al (17) found that the skin started to freeze at roughly -13°C and -9°C respectively. However, according to Danielsson (5) these are some 3-4°C too low because of thermocouple error. Another discrepancy is that Keating and Cannon (8) measured the skin temperature from an intracutaneous track with the finger precooled to low temperature. In the other studies the temperature was measured on the skin surface with a thermocouple, with the assumption that a "true" skin temperature was obtained. This procedure, however, introduces the same type of error as that found in the Siple and Passel WCI-factor.

Based on the assumption that the skin between cutis and subcutis starts to freeze at -1°C, the physical model produced skin surface temperatures that were very close to those measured by Wilson and Goldman (16) after these had been corrected for the errors mentioned above. It was found that the skin surface temperatures were linearly related to the frequency of finger frostbite (5). The results (figure 3) suggest that the risk of finger frostbite for those individuals tested, increase linearly from 0 to 100% as the skin surface temperature drops from -4,6°C to -8°C. This relation has been used as "human" anchor in the physical skin model, together producing the windchill frostbite risk model. The extended model, now including evaporative cooling and solar radiation, is based on the same risk - skin surface temperature relation. The assumption has been that type of heat transfer avenue is not important to the occurrence of skin freezing, only what skin surface temperature that is reached.

From the cumulative distribution curve any combination of skin temperature and risk frequency can be obtained. In the risk nomogram the three risk levels 5, 50 and 95% with the related surface skin temperature -4,8°C, -6,3 °C and -7,8 °C respectively have been selected.

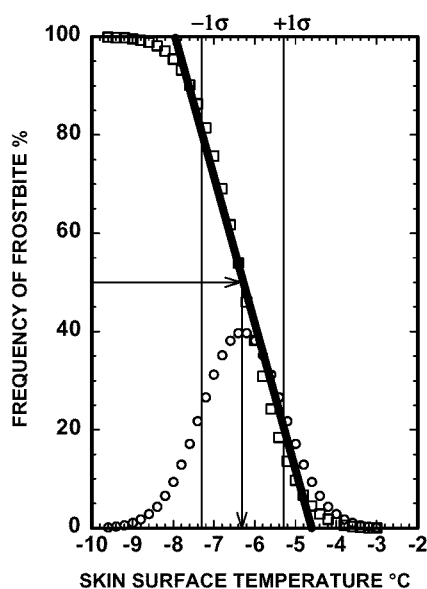


Figure 3. A standard normal distribution curve with a mean of $-6,3^{\circ}\text{C}$ and SD (σ) of 1°C . Related cumulative distribution curve closely follows relation between calculated steady-state skin surface temperature and frequency of finger frost bite. -1σ and $+1\sigma$ lines show skin surface temperature range where 68% of all frostbite cases can be expected for individuals who participated.

Fig. 4 shows what risk of frostbite a group of non-adapted people is exposed to for various combinations of air speed, air temperature, solar heat absorption on the skin when the skin is dry or wet. The risk curves derived for dry skin show a strong non-linear shape that basically mirrors the air layer thickness and its dependence on the air speed (c.f. fig. 1). At low air temperatures, around -30°C , an increase of air speed with roughly 1 m/s can be balanced by a 5°C rise in air temperature for the same frostbite risk. At -25°C a similar rise in temperature balances a 4 m/s increase in air speed and at -20°C the corresponding relation is 5°C versus around 12 m/s change in air speed. The risk curves for wet skin show similar shapes as the dry skin curves due to the close relationship between dry and evaporative heat loss (Lewis relation). The curves indicate that at air temperatures around -10°C or above, where the risk of freezing a dry skin is small, the risk becomes considerable great if the skin becomes wet. Bright sunshine combined with a high ground reflection value reduce the risk of skin freezing substantially. The solar radiation, absorbed by the skin can reach 1000 W/m^2 or more during the summer in Antarctic. At temperate latitudes, the corresponding absorption rate is considerably lower but $400\text{--}500\text{ W/m}^2$ can probably be reached on a clear day if the ground is covered by fine, fresh snow.

Validation

It is difficult to validate the risk curves because of ethical reasons. The amount of experimental data except those used for the development of the risk curves are very limited. The empirical data from outdoor activities often lack of important information. The climatic situation is often given as time-averaged figures and the relative air speed is sometimes estimated by experience or obtained from the standard 10 m level, often at some distance from the location of exposure. Nevertheless there are some unused data or implications that can be used for validation of the risk curves.

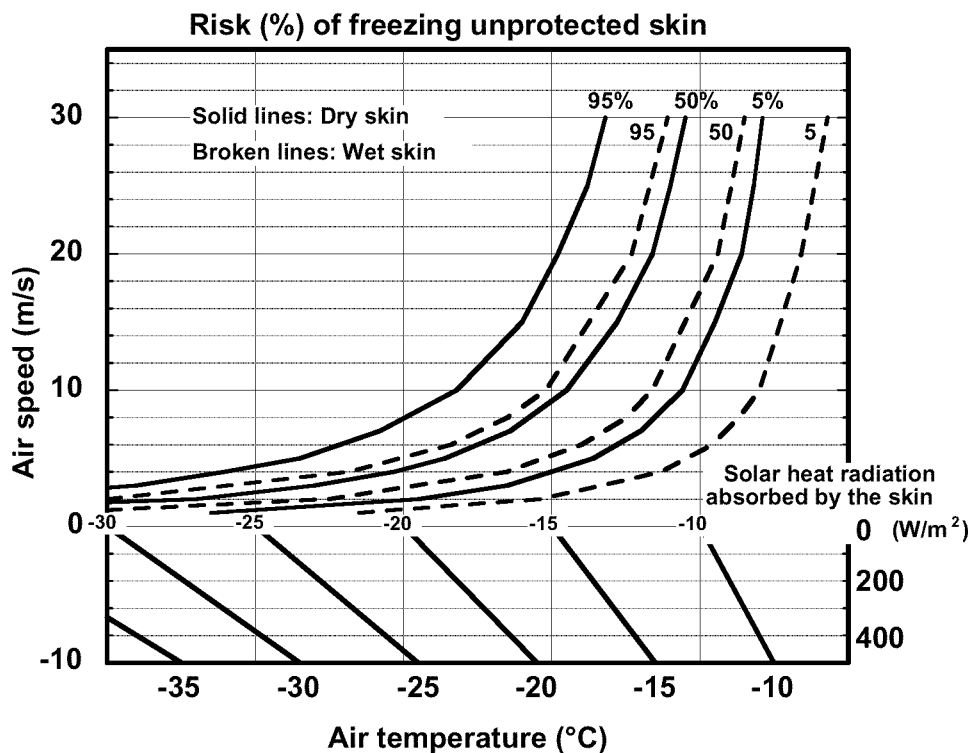


Figure 4. Risk of frostbite on windward side of a slim body part (diameter 2 cm) at various airspeeds, temperatures and solar heat absorption. Directions for use: From solar heat absorption go horizontally to air temperature. Then go vertically to air speed. Estimate risk of freezing the skin from solid curves in case of dry skin, else use broken curves (wet skin). Example: 200 W/m² of solar radiation absorbed by the skin and an air temperature of -15°C gives 50% risk of frostbite if the air speed is 13 m/s and the skin is dry. A wet skin increases the risk to approx. 90%.

Dry skin. Controlled experiments. Table 1 shows a comparison between predicted and observed frostbite frequencies from Wilson et al (17) and previously unpublished data of Wilson and Goldman (16). The data of Wilson et al (17) are given as average frostbite risks (45%) because the number of exposures under each climatic condition are not known. There seems to be fairly good agreement between predicted and observed data, except with some of the previously unpublished data (16). The reason for this can be that experimental conditions (these are unknown) are not comparable with those on which the risk curves are based. A change from 0% to 63% observed frostbite frequency as a result of only 2°C lower temperature at 10 m/s (16) supports the suspicion that other factors have been involved. But it should also be kept in mind that the controlled experiments rarely involved more than 6 or 7 subjects. So, a "random" variation in number of frost-bitten subjects of one individual causes a 15-20% change in "frostbite risk".

Table 1. Comparison between observed and predicted frostbite frequencies. * Average result; ** unknown experimental conditions.

air temp/air speed (°C/m/s)	observed frostbite frequency (%)	predicted frostbite frequency (%)	reference
-15/6,8	45	35	Molnar et al. (13)
-15/6,8	45*	30	Wilson et al. (17)
-15/9	45*	50	Wilson et al. (17)
-15/10	45*	55	Wilson et al. (17)
-12/10	45*	20	Wilson et al. (17)
-13,5/10	80	35	Wilson and Goldman (16)**
-17,5/4,5	0	30	Wilson and Goldman (16)**
-11,5/10	63	15	Wilson and Goldman (16)**
-9,5/10	0	0	Wilson and Goldman (16)**
-14,5/6,5	46	30	Hughes (Wilson and Goldman)(16)**

Outdoor frostbite injuries. A five-year review of the risk of cold weather injuries among U.S. soldiers in Alaska (1) shows that almost 40% of the injuries were frost-bitten ears and noses. Slightly more than 50% of the injuries were related to the feet and hands. It is reasonable to believe that the frequency of the frost-bitten ears and noses should depend on the windchill whereas the hand and feet injuries probably could be more related to the temperature only. However, the study indicates that the accumulated frequency of cold weather injuries was well related to the equivalent temperature (T_e).

Chandler and Ivey (1) found that the greatest increase, from 30% to 80% of all injuries (accumulated causality frequency) occurred at the equivalent temperature range -30°C to -40°C . They also found that more than 70% of all injuries occurred below $T_e = -29^{\circ}\text{C}$. The reference air speed 1,67 m/s was used for the T_e calculations. These results can, with some caution, be used for validation of the risk model. If the predictability of the nomogram is acceptable both for laboratory exposures as well as cold injuries in Alaska during very different environmental conditions including military activities, would make the nomogram fairly general. Table 2 shows the windchill chart (15) where the T_e -values are replaced by the risk value (assuming dry skin and no solar radiation) calculated for the same air speed and temperature. The marked cells in the chart correspond to the T_e -values ranging from -30°C to -40°C . The chart is stratified into three zones with ascending risk expressed as "Little danger" (upper left), "Increased danger" (middle) and "Great danger" (lower right). The table shows that the frostbite risk values, calculated for a slim body part ($d = 2$ cm) coincide fairly well with the marked cells showing when the accumulated frequency of cold weather injury increased from 30 to 80%. Taking consideration in that the accumulated frequency for a specific T_e slightly underestimates the actual risk frequency this could mean that the prediction value of the nomogram is still better than shown. The table also indicates that the risk zone classification "Little danger, Increased danger e.t.c. " seems to underestimate the risk as the change from 30 to 80% of all injuries occurred between "Little danger" and "Increased danger". The same comparison as in table 2 was done now assuming that the exposed body part was wider, with the diameter 15 cm as e.g. the head (Table 3). The table shows that risk predictions clearly underestimate the reported injury frequencies. The conclusion is that slim parts as nose, ears and fingers should be more exposed to injuries than wider parts as e.g. the face. This is confirmed by the reported data (1) as only 8% of the injuries were related to the face and other parts of the body.

Table 2. Windchill chart showing risk levels (%) for frostbite at various combinations of air temperature (°C) and air speed (m/s) assuming dry skin and no solar radiation.. The risk levels are taken from figure 4 and assume a slim body part as nose, ear, finger e.t.c. (diameter $d = 2\text{cm}$). The grey-marked cells denote T_e ranging from -30°C to -40°C (ref. air speed $1,67\text{ m/s}$). The upper-left zone refers to "Little risk", the middle zone to "Increasing risk" and the lower-right one to "Great risk" (15).

$v \backslash T_a$	-6,6	-12,2	-17,8	-23,3	-28,9	-34,4	-40	-45,6	-51
Calm	0	0	0	18	47	76	100	100	100
2,2	0	0	0	32	68	100	100	100	100
4,5	0	0	35	82	100	100	100	100	100
6,7	0	6	62	100	100	100	100	100	100
8,9	0	18	79	100	100	100	100	100	100
11,1	0	29	97	100	100	100	100	100	100
13,4	0	38	100	100	100	100	100	100	100
15,6	0	44	100	100	100	100	100	100	100
17,9	0	53	100	100	100	100	100	100	100

Table 2. Windchill chart showing risk levels (%) for frostbite at various combinations of air temperature (°C) and air speed (m/s) assuming dry skin and no solar radiation.. The risk levels are taken from figure 4 and assume a body part with a diameter of 15cm (the face e.g.). The grey-marked cells denote T_e ranging from -30°C to -40°C (ref. air speed $1,67\text{ m/s}$). The upper-left zone refers to "Little risk", the middle zone to "Increasing risk" and the lower-right one to "Great risk" (15).

$v \backslash T_a$	-6,6	-12,2	-17,8	-23,3	-28,9	-34,4	-40	-45,6	-51
Calm	0	0	0	0	0	0	12	29	44
2,2	0	0	0	0	0	6	26	44	62
4,5	0	0	0	0	24	47	74	100	100
6,7	0	0	0	18	50	79	100	100	100
8,9	0	0	0	35	71	100	100	100	100
11,1	0	0	12	50	88	100	100	100	100
13,4	0	0	21	62	100	100	100	100	100
15,6	0	0	29	74	100	100	100	100	100
17,9	0	0	38	85	100	100	100	100	100

Acclimation. Acclimation factor in terms of time stayed in cold regions has been reported to give fewer frostbite injuries. It is not clear whether the acclimation mirrors a more competent behaviour during cold exposure or if the reduced risk is an effect of well-documented physiological adaptation giving less susceptibility to frostbite. A guess is that both behaviour and physiology contribute to a lower risk. Massey (12) reported that during a controlled windchill exposure 74% of first year men on Antarctic developed frostbite whereas only 29% of the second year men. Candler and Ivey (1) also identified a higher risk for frostbite for the first year soldiers in Alaska, yet without reporting a more specific result. Applying the Massey-data to the risk nomogram, it suggests that people with more than one year of cold weather experience can endure twice as high air speed, or $2 - 5^\circ\text{C}$ lower temperature, as newcomers can for the same risk of freezing the skin.

Wet skin. Controlled experiments and outdoor experience have indicated that wet skin increases the risk of frostbite. Frostbite at "low" WCI is often associated with when there is snow in the air because of snowfall or strong winds (snowdrift starts at air speeds greater than 8-9 m/s (10)). The process responsible for the increased risk have not, as far as this author knows, been investigated. However, if the heat loss is roughly constant, the skin surface temperature falls continuously (laboratory situation). Outdoors, it may vary depending on the boundary air layer thickness which in turn results from factors as physical activity, wind direction, precautions taken to break the wind e.t.c. During such dynamic situations it is possible that the surface skin temperature passes through the freezing point. If the snow melts on the skin an evaporation process starts increasing the rate of heat removal from the surface. According to Massey (12) "skin numbness" came up to a much greater extent when there was snow in the air compared with no snow. He found that at windchill of 800-1000, snowdrift increased the numbness frequency with a factor of two whereas the increase was six-fold at windchill between 1000 and 1200. Skin freezing does not necessarily follow skin numbness, but a greater numbness index indicates lower tissue temperature, probably due to a greater cooling rate. However, these data can not be used for validation of the wet skin risk curves. Molnar et al. (13) studied the effect of controlled skin wetting. They found that water in epidermis caused skin frostbite in 6 out of 7 subjects (86%) whereas with dry skin frostbite was developed in only 3 cases out of 7 (43%). These results were obtained at an air speed of 6,8 m/s and the temperature -15°C. The risk curves for wet skin suggest 70% risk of freezing the skin whereas dry skin should only cause frostbite in 30 % of those individuals exposed. This study was performed with the skin quickly wetted (less than 30 s), a procedure that prevents the skin to be soaked with water. The same study revealed that freezing took place at slightly higher skin temperature when the skin was wetted. This finding is consistent with the physical model as water in epidermis lowers the insulation value. Keeping the skin freezing temperature and ambient air temperature fixed the surface temperature must rise due to the water in epidermis as long as the outer air layer insulation is unchanged. These mechanisms and its effects on the temperature distribution and heat flux can be simulated. The present model shows that the contribution from a dry epidermis to the total thermal insulation is very low meaning that a wet skin should only give a small contribution to the risk of freezing the skin, provided evaporation is prevented.

Solar radiation. Solar radiation reduces the risk of skin frostbite. This has been stated several times in the literature, primarily by members of Antarctic expeditions (e.g. Siple and Passel (14)). From heat transfer theories it is highly reasonable that if the cooling rate is slowed down by e.g. heat input from an external source applied to the skin surface, the risk of frostbite should be reduced. Yet, this effect has not, as far as this author knows, been shown explicitly in controlled experiments. So, the risk nomogram can not be validated strictly in this sense. The statement can only be supported implicitly. Chrenko and Pugh (2) have thoroughly described the contribution of solar heat radiation to the human heat balance. Based on observations at Maudheim, Antarctic by Liljequist (9) they showed that the direct solar radiation, on a clear day, is around 750 W/m^2 at an solar altitude of 10° and about 1050 W/m^2 at an angle of 40° . More relevant for an individual performing normal outdoor activities is the intensities measured on a vertical surface, e.g. the face. There the corresponding figures are 175 and 750 W/m^2 , respectively. Important is also the ground albedo when the it is covered by fine, fresh snow. The indirect radiation can be of the same magnitude as the direct one. Even when the sky is overcast the radiation can be up to 60% of the direct radiation on a clear day. This is because of the highly reflective snow producing multiple reflections between the ground and the clouds (in temperate latitudes this value rarely exceeds 25% (9)). So, the total radiation falling on a vertical surface, including sky albedo, is around 400 W/m^2 at the solar altitude 10° and as much as 1500 W/m^2 at 40° . Even with the face turned away from the sun the solar radiation is still about half these values. Assuming that the short-wave absorption of the skin is 65% the amount of heat absorbed by the skin ranges from about 150 W/m^2 at 10° solar altitude with no direct solar radiation to about 1000 W/m^2 at an altitude of 40° with direct radiation included. A rough guess is that these values should be reduce by 50% for temperate latitudes. In Antarctic, at -15°C and 9 m/s with no solar radiation the frostbite risk curves suggest about 50% risk of skin injury, a risk that is reduced to 5% if only indirect radiation reaches the body at an solar altitude of 40° . As this combination of air speed and temperature is rare during the Antarctic summer (18) skin frostbite should be uncommon. This is also in line with the experiences made. The effect of the solar radiation has also been expressed as equivalent rise in air temperature. Subjective opinions (e.g. 2) suggest that solar heat input may correspond to 5 to 10°C rise in air temperature. This is in line with the present model because the solar radiation intensities mentioned above correspond to a similar temperature range in the risk nomogram.

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